PEARL

The Journal of HEREDITY

FIVE GENERATIONS IN NINETY-NINE YEARS

(See Page 254)

The Differential Birth Rate
Genetics and Animal Disease
Are Plant Breeders Inventors?
Five Generations of Malformed Ears
One Generation in Ninety-Seven Years
The Genes Involved In Cancer Susceptibility

VOLUME 28 JULY, 1937 NUMBER 7

The American Genetic Association

An incorporated organization devoted to promoting a knowledge of the laws of heredity and their applicaiton to the improvement of plants, animals, and human racial stocks

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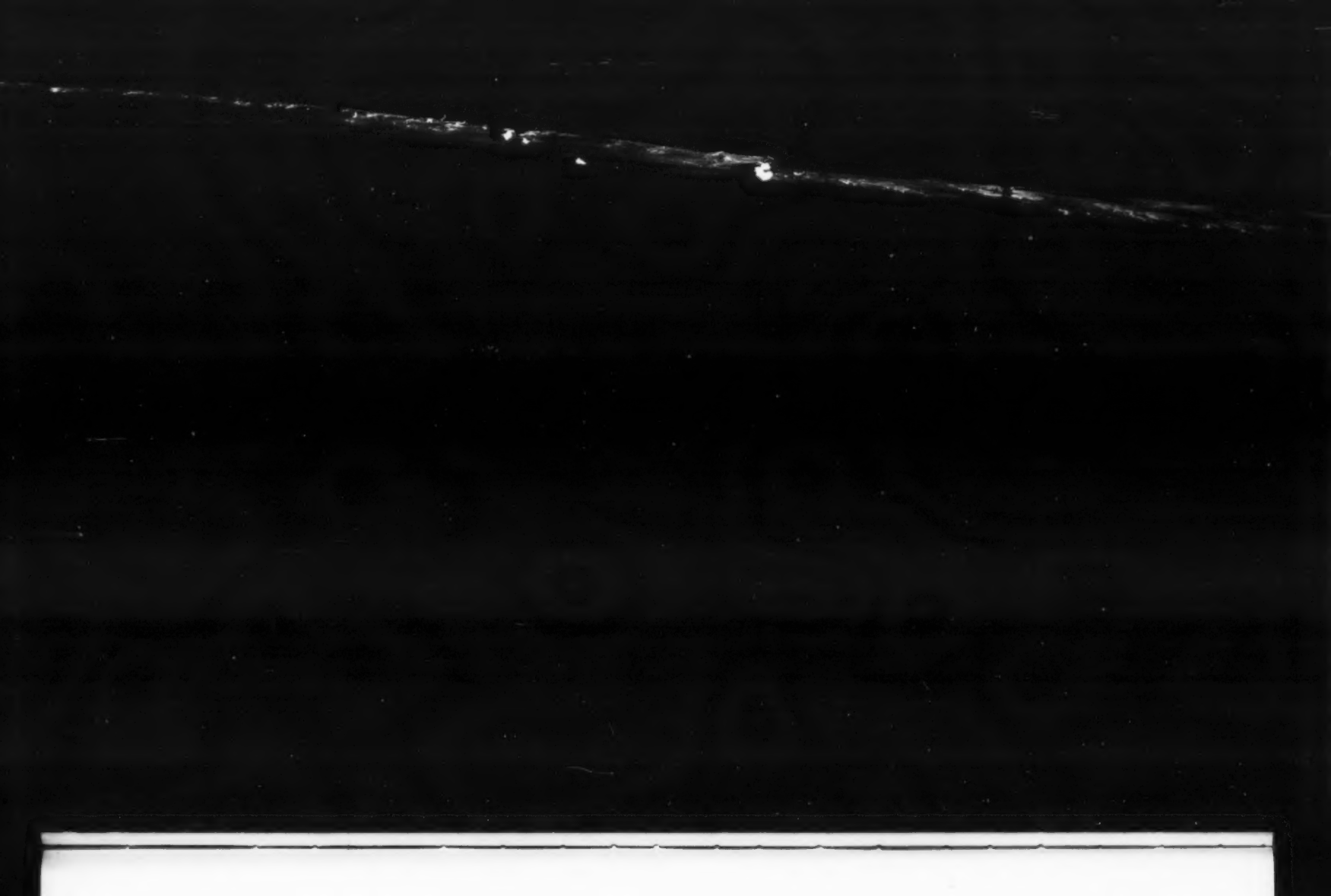

The

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TO SOME it will be tasteless to others it may be bitter, sour, sweet, or salty. These differences in taste reaction are inherited, inability to taste the substance apparently being transmitted as a Mendelian character. [A. F. Blakeslee and A. E. Fox, *Journal of Heredity*, March 1932 and *Proc. Nat. Acad. Sci.* January 1932.]

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Last year the "Taster test" served as an introduction to human genetic differences in many classrooms. The test is so easily made with the treated paper, and arouses such great interest that it appears to have a definite place in group demonstrations of such differences. These leaflets, size 3⅜" by 5½", are available in quantities for those who wish to use them for classroom or lecture purposes. On the back is printed an outline 4-generation pedigree chart. Sample on request. Prices: 10 for 25c; 50 for $1.00.

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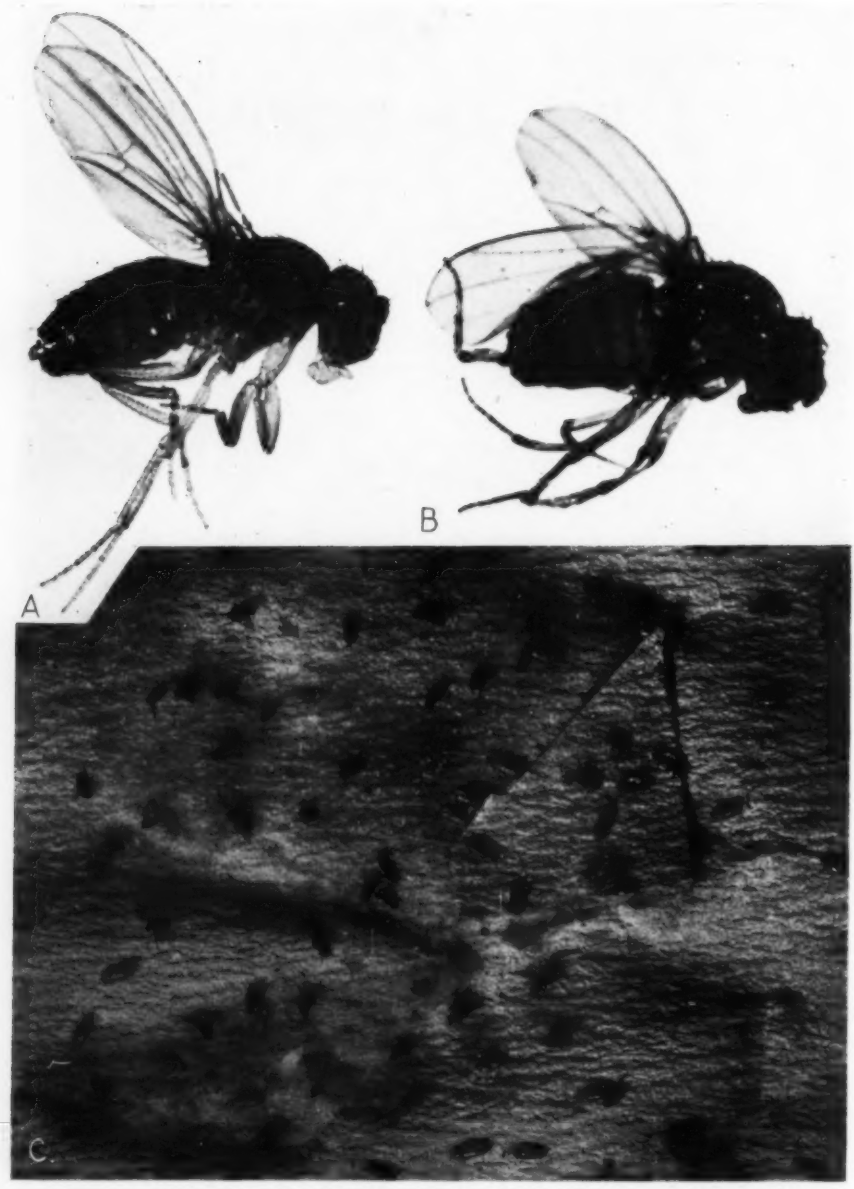


A LETHAL CHARACTER AFFECTING THE LEG JOINTS OF DROSOPHILA

Frontispiece

A, normal fly; *B*, fly suffering from focal melanosis which produces melanotic lesions of the joint between the femur and tibia. The dark joints of the legs can be seen easily in the photograph. *C*, a hatching brood of fruit flies segregating for this sex-linked recessive gene. The flies emerge from the pupae cases but cannot move normally and die. Many lethal and semi-lethal genes are known in Drosophila at the present time. Some of them become effective early in embryonic development, and others only show their lethal effects after adult development has been reached.

CONTRIBUTIONS OF GENETICS TO UNDERSTANDING OF ANIMAL DISEASE

JOHN W. GOWEN*

DISEASE in domesticated animals has been viewed quite differently from disease in domesticated plants. Due to the prestige of Pasteur and Koch, or perhaps more properly to the lieutenants in the victorious bacteriological army, it came to be believed that when the means of infection and vehicles of infection were identified the problem of an outbreak of herd illness was solved. Diseases were to be treated by campaigns of eradication, or when that would not do, by the arts of vaccination or immunization. The host was but a container having no particular attributes of its own. The successes of this program are known to all, yet this approach was not without heartache, as when Shiga says, "Discovery of dysentery bacillus had stirred my young heart with new hopes of eradicating the disease and saving the sufferings of about one hundred thousand cases that occurred at the time. Thirty-eight years had passed since this heart-throbbing event happened. Many thousand people still suffer from it year to year, and the hope that once had been thought would bring new light had disappeared as a dream of summer night."[22]

Disease of plants had another tradition. As early as 1797 the rust of wheat was recognized as due to a specific agent, and varietal differences were established by 1840. The host part in the disease syndrome became accepted thought, and breeding to collect desirable germ plasm within a variety an established practice. As an animal geneticist I feel quite humble and can truly say of my associates on the plant side that they are the better half in the development of disease resistant strains.

In the nascent state of the animal side it is my purpose to emphasize genetics as penetrating the disease problem and to portray what the development of disease resistance may entail. We would, I presume, agree that disease results from the interplay of three groups of factors: (1) the genetic constitution of the host, (2) the disease producing entity, the pathogen, (3) the environment. Any one or any combination of these causes may bring about disease.

Gene Disease

The yellow mouse case[2] has become an established example of the fact that the internal organization of an embryo, even to one of its single genes, is capable of ending the life it had a part in beginning. The case did not tell why the yellow organism died; it gave only the result. This void has been partly filled by lethal or semi-lethal genes which do not kill their host at as early a stage in development. Focal melanosis,[7] due to a single recessive sex-linked gene, causes melanotic lesions of the femur-tibia junction. Affected flies crawl out of the pupa cases but, being unable to move normally, die. A closely prescribed lesion has resulted in death. (See Frontispiece.)

Cases like those cited above, in which specific genes alone are responsible for a certain pathological condition, are known for every species utilized in genetic research. Records show that

*Presented at a symposium of plant geneticists and pathologists at Atlantic City December 31, 1936. The other contributions to this symposium, by Prof. E. C. Stakman (plant diseases) and by Dr. H. A. Rodenhiser (segregation and mutation in plant pathogens), have been promised for later publication in the Journal.—ED.

observed deaths are often centered about critical stages in the developmental processes when the organism changes from one type of life to another, that is, hatching in oviparous, or birth in viviparous species. But as the work of Nishikawa[18] on lethals in the silkworm shows, the lethal genes may have their effect in early segmentation stages, in retarded embryonic growth, or even 15 days after hatching, and yet have the deaths observed late in development.

Lethals in cattle are of particular interest. Breeders favoring Dexter cattle[1] have fixed as the fundamental character of their strain a character dependent on a gene that is lethal when homozygous. Wriedt and Mohr[17] have studied two lethals in the Swedish Holstein breed. Each lethal entered the breed through a popular and different sire. As each sire was heterozygous and appears an ancestor in two-thirds of all pedigrees, the lethals are widely scattered within the breed, so widespread that some herds have had difficulty in maintaining their full numbers because of the deaths due to them. Owners of herds suffering from lethal deaths have searched for stock unrelated to theirs from which to purchase bulls, only to find when they had bought a bull that he often carried both lethals. Breeding methods designed to free the breed of these lethals specify that bulls selected as breeders should have 20 normal calves in matings to their own daughters before being used on a large scale. With bulls which commence breeding at 2 years, this requirement means that a bull would be 7 years old before he is put in general breeding. Should this requirement be made a practice, 14 out of 15 heterozygous bulls would be detected before they would have spread their defective germ plasm to the rest of the breed.

Besides the lethals mentioned, others cause the skin of the legs and the mucous membrane to be so defective as to allow bacterial invasion followed by septicemia and death;[13] the skin to be hairless; the extremities to be amputated; the skeleton to be foreshortened; the eyes to develop cataract of the stellate type,[6] etc. A dozen or so lethals are known for cattle. Other species have similar numbers, although the number is apparently limited only by the amount of study which has been devoted to the particular group.

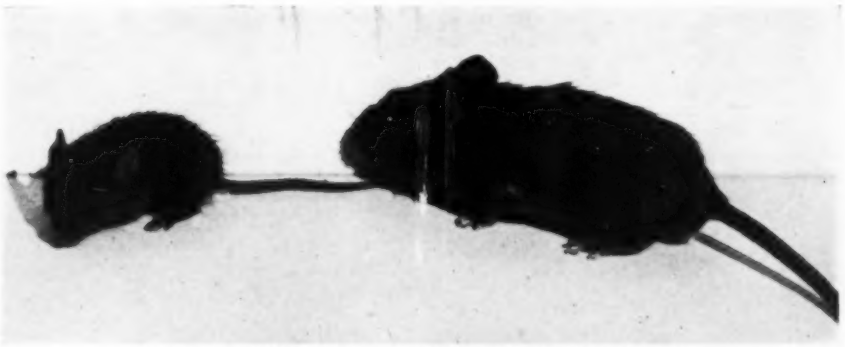
A GENETIC LETHAL SUBJECT TO SURGICAL CORRECTION

Figure 1

This is an example of an hereditary disease (conditioned by a recessive gene) which may be corrected by surgical interference. Imperforate vagina in the mouse causes eventual death by retention of uterine secretions. The hugely distended horns of the uterus show how this inheritance acts. Operative removal of the obstruction prevents the lethal effect and results in normal fertility in affected individuals. While this lethal effect can be "cured," the development of a strain dependent on such surgical intervention proved impractical even under laboratory conditions.

In addition to overt anatomical defects, genes may affect the well-being of the animal, as in basal metabolism or vitamin B utilization in the domestic fowl,[10] or vitamin D in the rat.[9]

Following the trend to alleviate animal disease by some method of treatment, the question may be asked, are hereditary diseases necessarily incur-



A SEMI-LETHAL DUE TO GENETIC GLAND DEFECT

Figure 2

Dwarf mice due to inherited defect of the pituitary gland may be rendered normal in appearance and function (except for sterility in females) by transplantation of normal pituitary tissue into the dwarfs. The lethal effects of this gene can be masked and the strain perpetuated through affected males by surgical means, but the story of such a strain if palliative measures are withheld would be short and tragic. The practical implications of such a situation are brought close home when we consider the possible effects of fostering a higher birth rate among diabetics through use of insulin. In some families there is evidence that diabetes is due to an inherited deficiency of the panceas. The perpetuation of such gland-deficient strains by insulin therapy would definitely foster the development of a strain of human beings which could not survive without insulin treatment. The wisdom of such a course may be questioned on humanitarian and other grounds.

able? Surgical interference has shown some types of hereditary defects capable of repair. Imperforate (vagina) in the mouse prevents reproduction, causes acute distress due to retention of uterine secretion, and ultimately leads to death. Surgical removal of the obstructing membrane allows normal life and reproduction. (See Figure 1.)

Physiological defects due to inheritance may also be alleviated. As Detlefson[5] and de Aberle[4] showed, mice homozygous for lethal anemia seldom survive more than a week from birth. The injection of the whole blood into such mice as I have shown,[10] extended this period even though it did not fully compensate for the defective germ plasm. A similar, but more successful case where the deleterious effects of a gene were eliminated is Snell's[24] dwarfism in the mouse. The dwarf animals are small, sterile, and show thyroid, adrenal, and reproductive repression. Basal metabolism is reduced to one-half the normal. Transplantations of pituitaries of normal mice into the dwarfs were shown by Smith and MacDowell[23] to cause a resumption in growth and a return of the thyroids, adrenals, and reproductive tract (save for the female) to normal. But the primary gene effect is not altered, the eosinophil cells of the pituitary do not come back. The replacement therapy has demonstrated the possibility of repair in hereditary disease and shown first class evidence of the physiological utility of the eosinophil cells. Yet in turn it presents the problem of the further perpetuation of defective inheritance in much the same form as the problem is coming to the fore in diabetes with the introduction of insulin. (See Figure 2.)

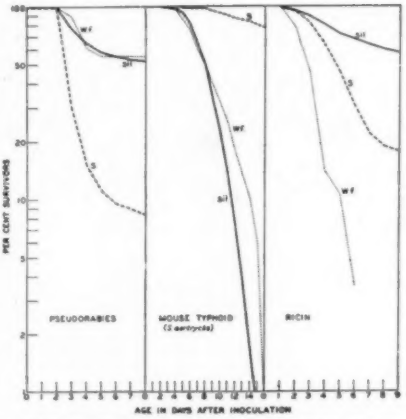
Infectious Disease

Infectious disease with its accompanying pathogen introduces other elements into the reacting system. The pathogen, as well as the host, has its characteristics prescribed by its ancestors. Instances where susceptibility or resistance to a disease turns on a single pair of genes are rare. The epidemic

liver disease of mice, first studied by Tyzzer,[20] is possibly an example. The pathogen involved is a long slender bacillus, *B. piliformis,* with special growth preferences. It will attack one species, the Asiatic mouse, but our common mouse is resistant. Resistance segregates as a dominant.[12]

A similar instance is found in the guinea pig. Through the accident of using large numbers as a source of blood complement Rich[19] isolated a strain deficient in this material. Breeding experiments showed the character, full vs. deficient, to depend on a single gene pair, the deficient complement being recessive. In a spontaneous epidemic in which *B. suisepticus* appeared in these stocks, the complement-full pigs often developed lesions of the liver, spleen, and peritoneum, but seldom died. A large part of the low-complement pigs, on the other hand, died so suddenly that they did not even develop characteristic lesions. The focus of the epidemic was the deficient-complement pigs. An experiment to test this matter further was performed. One hundred pigs of each group were inoculated with living *B. cholerae suis* under similar conditions. Of the 100 complement-deficient pigs, only 23 survived at the end of 48 hours; of the normal full-complement pigs, 80 were alive. A single gene pair had markedly affected the resistance through changes in a serum chemical, complement.

Unfortunately but few diseases have so simple genetics as Tyzzer's liver disease or Rich's complement deficiency. But susceptibility or resistance may be markedly influenced through proper breeding. Speath[25] on pneumonia, Irwin[14] on rat typhoid, Roberts and Card[20] on bacillary white diarrhea, Lambert and Knox[15] with fowl typhoid, Schott[21] (aertrycke) and Webster (enteritidis) with mouse typhoid, have all demonstrated genetic differences in disease resistance. Rise in antibody titer from one exposure to disease, as in ricin poisoning, also has a genetic basis. Examples of host resistance are known for the whole range of patho-



DIFFERENCES IN REACTION TO THREE LETHAL AGENTS

Figure 3

Three strains of mice exposed to a virus disease (pseudorabies), a bacterial disease (mouse typhoid), and an antigenic poison (ricin), show that each of these strains has a specific reaction to each of the lethal agents. The *S* strain was most susceptible to pseudorabies, least susceptible to mouse typhoid, and intermediately susceptible to ricin. The genetic constitution of the strain determined its reaction to a given infective or reactive agent.

genic agents, bacteria, fungi, protozoa, helminths, viruses, and chemical poisons. In fact, so far as I am aware, *no investigator who has adequately sought inherited host differences in disease response has failed to find them.*

Genetic constitution for disease resistance could conceivably be a single over-all character which enabled the organism to resist a number of diseases, as postulated in early medicine, or a composite of many independent characters each of which is capable of causing the individual to resist one disease, as present day genetic thought might expect. The fortuitous combination of these characters could make animals resistant to many or few such agents. Data taken from the work of my former colleague, Dr. Schott, and myself on the reactions of inbred lines of mice to different pathogenic agents are available for this comparison.

Three different strains were tested for their reactions to three diseases:

mouse typhoid, a bacterial disease, by Schott[8]; pseudorabies, a virus disease, and ricin, an antigenic poison, by Schott and myself. The survival rates for days after inoculation are placed on the arith log grid. The three figures for the different diseases have this in common. Each disease has strains of mice which are quite susceptible to the diseases and strains which are quite resistant. The pseudorabies plot shows that the *S* line is quite susceptible while the *Sil* and *W.F.* lines are both resistant. For mouse typhoid the lines are reversed; the *S* stock is resistant, whereas the *Sil* and *W.F.* strains are quite susceptible. The resistance toward ricin completes all possible combinations of resistance and susceptibility. The *Sil* line is most resistant, the *S* is susceptible, and the *W.F.* is nearly wholly susceptible. The genetic constitution for resistance is evidently specific for the material and strain. Constitution in disease resistance is, like most other characters, dependent upon the interaction of specific genes rather than upon an over-all type of reaction. (See Figure 3.)

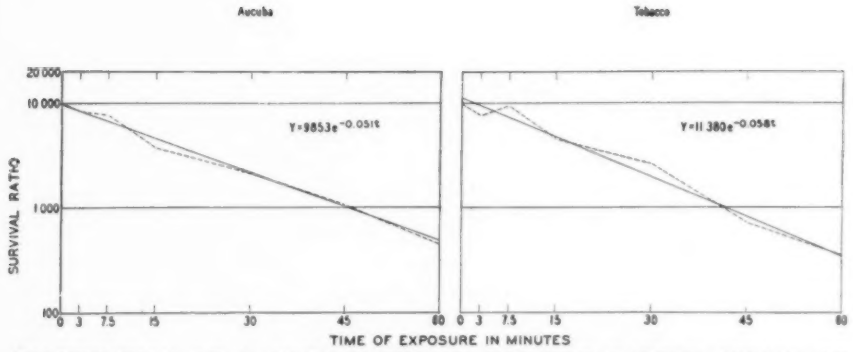
Variation in Pathogens

We have sketched the effect of variations in genetic constitution of the host on disease. Suppose we reverse the study and examine the effect of genetic variations of the pathogen. In examining these facts, may I call to mind the rapidity with which these organisms reproduce, eight hours being sufficient for the colon organism to grow to ten million, or enough organisms to allow one mutation for each gene carried by this bacillus. The effect on the adaptability of such large populations even though the rate of change is low was strikingly shown in 1887 by Dallinger and Drysdale[3]. Starting with a flagellate having a temperature requirement of 60° F., they were able to raise its requirement to 158° F. in a period of 7 years. The change in temperature adaptation was not a continuous process but rather a series of discrete steps unequal both as to length of rise and to generations which elapsed between rises. Similar changes were noted in the sugar reactions of colon bacilli by Massini, Stewart, and others. The results are favorable to the interpretation that the observed changes are the result of hereditary reorganizations of the nature of mutations. Similar alterations of virulence occur. The pneumococcus mutates from type I to type II in the presence of antiserum of type II, or from type II to type I when grown in the presence of type I serum.

Similar changes take place on host passage. Wellhausen[31] introduced a pure culture of *B. stewartii* into different strains of inbred corn, some of which were susceptible and some resistant to the disease. Repeated transfers of this organism in the resistant host increased the morbidity and mortality of the corn plants until it became stable for this strain. Passage of the bacteria through the susceptible corn strain somewhat decreased the observed mortality of the corn plants and stabilized the bacteria isolated from these plants at a lower level of virulence. Virulence in the first case, avirulence in the second, became characteristics of these two strains so long as the bacteria were maintained in the particular strain of corn.

Two relatively pure races of *B. stewartii*, differentiated for virulence, had been produced from the one parental population: one pathogenic, at least to some degree, for all races of corn; the other slightly pathogenic. To complete the picture these strains were again split into two. The half of the virulent strain on the resistant host retained its virulence; the other half after several passages in the susceptible race had lost virulence. The susceptible strain, on the other hand, had after several passages in the resistant host, gained virulence and it was as good as the virulent strain first established. On the susceptible race of corn the strain merely retained its pathogenic property for the susceptible race.

You may say that this is an old story, simply the gain of virulence through host passage. But there is mysticism in this older view. The experiment out-



INACTIVATION OF A MUTANT STRAIN AND THE PARENT STRAIN OF TOBACCO MOSAIC VIRUS BY X-RAYS

Figure 4

Above is shown the effect of X-rays on common tobacco virus and on aucuba, a mutant form derived from the common tobacco virus. The close parallel in the inactivation rates of the two viruses exposed to energy of this high frequency suggest similar physical properties in these two viruses. The further similarity of these effects to those observed in genes have equal interest. Dotted lines actual data, solid lines fitted curves.

lined tells the story better. It shows definitely that host passage in itself does not increase or lower virulence. It requires plants of definite genetic constitution to accomplish this result. It suggests the step-like process of mutation reacted upon by selecting agencies whether stringent or lax as the basis of changes in virulence. It shows virulence to conform to the attributes of genetic characters.

Strain resistance to tumors introduces yet another possible genetic factor in disease. The appearance of tumors has been postulated as due to simple gene inheritance, to somatic segregation or mutation of a body cell to one of unlimited growth, or to viruses which unleash the cell's growth possibilities. Without entering the controversial question of whether any or all of these hypothesis may account for tumor formation, let us examine the hypotheses for their genetic significance. The uncomplicated gene effects in disease have been discussed. Somatic segregation while infrequent occurs often enough so that all the cells of any one organism of the size of a fly are not necessarily identical genetically. The segregated cells, as Demerec has shown, are frequently not viable, or, as I have shown, may cause morbidity and death with certain environments. Somatic segregation may be governed by genes, as the work of Stern demonstrates[28], thus furnishing a mode of inheritance.

The somatic mutation hypothesis has a like interest. Papers presented at this meeting suggest that genes as well as external agencies may bring about mutation in other genes,—a fact which could go far in explaining the occurrence of tumor and non-tumor strains of mammals. Chemical agents, hormones, viruses utilized as initiating agents for tumors, are significant to us as the physiological effects they produce may put them in the category of agents effecting somatic segregation, aberrant mitosis, or gene mutations.

Viruses

Viruses as possible agents to unleash the cell's growth possibilities are of particular interest. For instance, tobacco-mosaic virus is composed of particles the size of which is estimated to be of the same order as that of genes. The viruses also resemble genes in other respects: both are incapable of reproduction outside of living cells; they produce similar effects, as, for instance,

variegation or mottling, in plants; and they are, under natural conditions, capable of mutating to new forms which retain the ability to reproduce themselves. The virus differs from genes in being able to move from cell to cell and in being capable of inoculation into the cells of healthy plants.

Investigations by Dr. Price and myself[11] have shown viruses to have further gene-like attributes. They are inactivated by both ultraviolet light and x-ray, the quantitative relations being similar to those observed for genes. It is thus possible that we have entities with properties comparable to those of genes which may be isolated for study. The work of Woods[32] and the researches of Vinson[30] in passing virus through chemicals inimical to life and suggesting protein structure, of Stanley[27], Wyckoff[26], and others on crystallization and structure, give an idea of the possibilities. (See Figure 4.)

For purposes of further discussion I will categorically present the results: (1) Genes normal to a species may, by mutation, cause physiological and developmental processes so abnormal that death or lasting disability results. (2) A proper understanding of hereditary disease may offer a means by which it may be alleviated. (3) The segregation of specific genes for susceptibility or resistance to disease initiated by pathogens may be responsible for immunity, morbidity, or mortality within the exposed population. (4) Similarly the segregation of genes for pathogenicity may make a pathogen virulent, mildly invasive, or innocuous. (5) The balance in the proportion of the genes making up the body may become the direct etiological agent of a definite pathological syndrome causing death or greatly reduced length of life. (6) During development the body, due to its inheritance, may become a mosaic of cells, some normal, some abnormal, and the morbidity and mortality increase directly with the proportion of defective cells in the mosaic. (7) The viruses as possible gene-like substances offer opportunities for the study of the genetic basis for the diseases they produce.

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Age Limit of Male Fertility

THE recent death of a North Carolina farmer, George Isaac Hughes, at an age of 97 (announced by the Associated Press on May 20), closes a unique career in long-term reproductive achievement. Father of sixteen children by his first marriage at an age of 32, Hughes married a second time at 95, and became the father of two children before his death. The case was investigated in 1935 by three physicians shortly after the birth of the first child, and the exhaustive medical examination made at that time left no doubt that this modern Abraham was physiologically able to be the father of children (*Jol. Amer. Med. Assoc.*, Nov. 2, 1935, P. 1423). "The spermatozoa were of the average size, and only 65 per cent of the customary number were present. Their conformation was entirely normal, and their motility was very great."

The *Journal* article does not identify the subject by name, but the identity in location (Bern, N. C.) makes it quite certain that the subject of this investigation is the man who has just died. The information below is from the article cited above.

Born in 1840, Hughes' boyhood was spent on the farm, and while he was in the Confederate army during the Civil War he was a miller in the Quartermaster Corps, and saw no active service. He married in 1872, and his first wife, who lived until 1925, bore him sixteen children, of whom six were living in 1935—the oldest, 63, the youngest, 33. He had 38 grandchildren at that time.

In 1935 Hughes married a widow of 27. The first child of this union was born in October, 1935, the second child in May, 1936.

The physical examination reported in detail in 1935 showed a rather remarkable condition of physical excellence considering the very advanced age of the subject. The "senile appearance" of the subject, whose teeth and eyes had passed their prime, was compensated for by excellent heart and lung action, a sound circulatory system, an excellent musculature, normal mentality and normal sensory reactions, of unusual excellence considering the advanced age, — "mentally the patient was entirely normal, his general behavior and cooperation being excellent; his emotional state was euphoric, and his orientation, memory, intelligence, and judgment were very good."

The case is unique in representing by far the greatest age at which male fertility has been verified by a competent medical examination. —R. C.

HEADED FOR THE LAST CENSUS?

II. The Differential Birthrate

GUY IRVING BURCH

(Continued from the June issue)

DURING recent years a great deal of scientific data concerning the quality of the American people have been accumulated. Much of this material concerns what is called the "differential birth rate," that is, differences in birth rates or rates of reproduction among economic, social, racial, occupational, educational, rural and urban groups in the population. Furthermore, during the past forty years, and especially during the last *twenty* years, scientists in many countries have pieced together psychological tests which have proved increasingly efficient in measuring man's "natural" intelligence even at a very early age.

About the time the United States received its first big wave of immigration in the middle of the nineteenth century, this country, like many industrial nations in Europe, began to experience a marked difference in birth rates among economic and social groups. At the World Population Conference at Geneva in 1927, the eminent British biologist, Professor Julian Huxley, said "it appears that the present differential fertility was unknown a hundred years ago." It is significant that Malthus, the founder of population theory, who lived during the first half of the nineteenth century, said little or nothing on this subject.

As late as 1850 in countries of Western Europe the upper social strata were reproducing at a greater rate than the lower strata, according to Professor S. J. Holmes[32] of the University of California, who quotes a number of authorities to this effect. Well-to-do farmers, he says, married before workers without property. Apprentices in trades seldom married until they attained a more or less assured position. Peasants were not permitted to expand their numbers freely by migration to cities, and pressure of population upon the land held down the size of their families. Modern sanitation was hardly known and the death rate among the poor and ignorant was terrific. Among the successful types there was little family limitation. With the early development of factory work on a large scale, about the middle of the last century, there came a great demand for cheap industrial labor which broke old ties and customs. The great emigration to America of the lower economic and social classes also relieved population pressure in European countries in these classes and left room for an increase of their birth rate. These factors had a tendency to increase the birth rate of the groups which supplied cheap labor in Europe and the immigrants in this country also had large families here. At the same time pioneers like Pasteur and Lister had revolutionized medical practice and increased medical efficiency began to lower the death rates of the lower classes, while the upper classes began to practice family limitation on a large scale.

Detailed statistics concerning birth rates and death rates for this country as a whole do not reach back to the middle of the last century but, according to Dr. Frank W. Notestein,[33] a recognized authority on American Vital Statistics, much the same thing was happening here as regards class difference in reproduction as was happening in England. Between 1840 and 1880, the average number of children among English landed gentry, according to Professor Holmes, dropped 50 per cent,—was cut in half. At the same time A. J. Lotka,[34] statistician of the Metropolitan Life Insurance Company, tells us that the general birth rate of England rose from 32.3 per thousand in 1841-45, to 35.3 in 1875-80.

Last year the Population Association of America cooperated with the American Academy of Political and Social Science in preparing the most authoritative and extensive work on American population that has yet been published. Dr. Notestein was selected to write the important chapter on "Class differences in Fertility." This authority presents figures to show that since 1855, miners, agricultural workers, and unskilled laborers have been rapidly outbreeding, and at an increasing rate, the upper and middle economic and social classes. The implications of these trends he hints at in the following words: "Today everyone who thinks about this matter at all agrees that the systematic recruiting of the nation's population from the working and agricultural classes may have an important bearing on the quality of the future population."

This point is brought out quite clearly by Dr. Frank Lorimer, now technical secretary of the Committee on Population Problems of the National Resources Committee, and Mr. Frederick Osborn, research associate in Anthropology of the American Museum of Natural History, in their authoritative book, *Dynamics of Population:*

> Some large groups are reproducing so rapidly and other large groups are so diminishing, that the surviving children of a million women of child-bearing age now living in some groups will be twice as numerous as the surviving children of a million women in some other groups. Such an extreme differential, if continued for three generations, would cause the descendants of the first group to be sixteen times as numerous as the descendants of the second group.[35]

Three generations are about one long lifetime. Thus some people now living will see this great shift in the composition of our population *if* the present trends in the "differential birth rate" do not change.

Let us now see what groups are over- and under-producing themselves. From Government statistics the above authorities "estimated that mothers with husbands in the unskilled laboring group were having over 15 per cent more children than would suffice for family replacement, whereas mothers with husbands in the professional group were having only about 75 per cent of the number needed for family replacement."[36]

Whether we study economic, social, cultural, occupational, educational, intellectual (as measured by intelligence tests), rural, or urban groups in this country, we find that the lower strata in each group have the largest families and that the higher strata have the smallest families. That this can have no bearing on the future of our country is inconceivable.

Intelligence Tests

The psychological intelligence test as it has been developed since the World War (and no doubt will be further perfected) is a powerful weapon with which to fight national decay. It is also a power that breaks through all economic and social barriers which have heretofore hindered the development of deserving children in poor families.

Intelligence scores are not calculated or classified like school marks.[37] Where school marks run only to 100, the limit of intelligence scores or I. Q.'s may in extremely exceptional individuals run as high as 200. Therefore, I. Q.'s ranging between 90 and 110 are merely average or "normal" intelligence. This is the class in which the great majority of the population is found. Scores of 150 or over are very exceptional and suggest genius. Individuals considered "dull" and "backward" by school teachers and in every day life are usually found in the 80 to 90 classification. Between 70 and 80 are generally found persons bordering on feeblemindedness, and definitely feebleminded persons or mental defectives are found with I. Q.'s below 70. Between 50 and 70 are found the so-called "morons" (high, middle, or low grade). Imbeciles are in the grouping from 25 to 50, and those below 20 or 25 are classed as idiots.[37] Of course, these are more or less arbitrary classifications used primarily for the benefit of school teachers and the exchange of knowledge between professional workers interested in measuring intelligence.

With this classification of intelligence scores in mind let us briefly consider some of the information that has been found concerning intelligence and size of families. Studies of clinic and school data concerning children in Bridgeport, New York City, Englewood, St. Louis, and vicinities,[38] show that children with the lowest intelligence scores (I. Q's below 60) came from families which averaged nearly six children. The next lowest group, the feebleminded (I. Q.'s 60 to 70), came from the next largest families, which averaged nearly five-and-a-half children. The borderline feebleminded children (I. Q.'s 70 to 80) came from families which averaged more than five children. The "dull" and "backward" children (I. Q.'s 80 to 90) came from families that averaged more than four-and-a-half children. Those considered "normal" (I. Q's 90 to 110) came from families that averaged less than four children. Those children reckoned of superior intelligence (I. Q.'s 110 to 130) came from families that averaged a little over three children. Lastly, the children of very superior intelligence, suggesting genius, (I. Q.'s 140 and over) came from families that average less than two-and-a-half children.

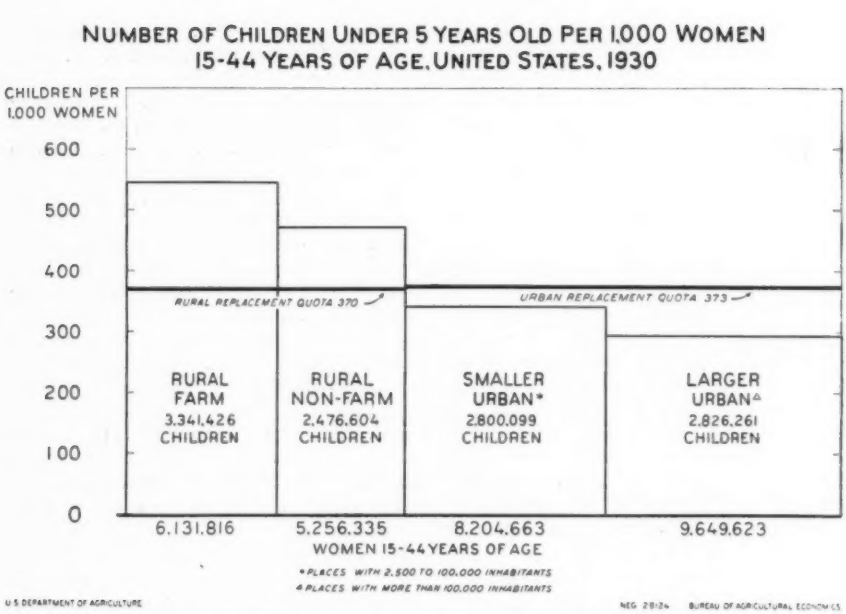
These differences in size of families among intelligence groups is made even greater as far as population growth is concerned when we take into consideration that the business and professional groups where the highest I. Q.'s are generally found contain from one-fourth to one-half more *childless* marriages than groups in the unskilled and farming occupations, where on the average the lowest I. Q.'s are found.[39]

Other studies are presented by Lorimer and Osborn which indicate that each group with intelligence scores below 100 (and 100 might be considered average for the country) is growing larger and each group with scores over 100 is growing smaller. In one generation (a period of about 28½ years), the highest I. Q. group declines more than 12 per cent, while the lowest intelligence group (I. Q.'s 50 to 60), who are definitely feebleminded, increases about 9 per cent.[40]

It may be thought that better diet, education, or better environmental conditions generally will compensate for this downward shift in intelligence of our people as measured by psychological tests. Dr. R. B. Cattell,[41] who has recently surveyed the situation in England, holds that the facts discovered do not verify this optimistic assumption. Children brought up in the same environments, such as in orphanages, show as great difference in intelligence as other children. Groups of children given intensive education and special feeding show the same rate of intelligence growth as do other groups. Identical twins (which have the same heredity) have practically the same intelligence scores, and even when reared apart from infancy no marked difference of I. Q.'s arises. Newman and his co-workers[42] in a study of nineteen pairs of identical twins reared apart found an average difference of eight points as compared with an average difference of about five points for fifty pairs of identical twins reared together.

In general, intelligence seems to be not very drastically modified by the range of environment encountered in the United States. We do not know of any environment that will make a genius out of a genetically feebleminded individual. The reverse of this proposition appears probably also to be true if we exclude pathological conditions such as disease and birth trauma.

It may also be thought that parents with low intelligence may give birth to children more intelligent than they are. This sometimes happens, as does the converse—that very intelligent parents sometimes give birth to much less intelligent children or even to idiots. Neither of these occurrences constitute exceptions to genetic theory when multiple facors are involved. But as a rule the Biblical saying, "we do not gather figs from thistles," appears to hold when we consider the inheritance of mental ability. For example Cattell, in testing groups of parents with I. Q.'s ranging from the highest to the lowest classification, found without exception that the children were in the same general I. Q.



HOW CITY AND COUNTRY MEET REPLACEMENT QUOTAS

Figure 5

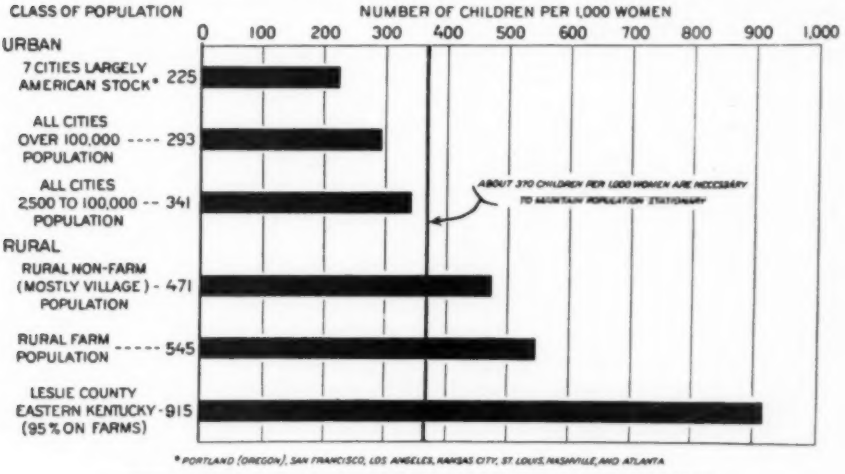
In the rural United States in 1930 were 5,818,030 children under five, and 11,388,151 women between 15 and 44 years of age. In the cities were about the same number of children (5,626,360), but a third more women (17,854,286). As a consequence the United States is reproductively "in the black" in the rural districts, but "in the red" in the cities. The significance of these facts in determining what kind of a people future Americans will be depends on the extent to which migration from the country to the city is a selective process.

classification as their parents, and that this was true whether or not the children lived with their parents. For example, children resembled their *real* parents in intelligence far more than they did the foster parents by whom they were reared from infancy.

The Urban Trend

Another important element in determining the vitality of the American people is that of their changing living conditions. It is generally known that during recent times there has been a great movement of people from the country to the cities. According to Department of Agriculture figures[43] nearly 27 million persons have left farms for cities, towns, and villages since 1920. It is true that nearly 20 million arrived at farms during this period, leaving a net balance of about 7 million in the urban centers. But the people who moved from the country to the cities are thought to be quite different from those who moved to farms. Those going to the urban centers are usually young, energetic, and relatively intelligent, usually the kind of people who are in the most favorable ages for bearing and rearing children and also the type which would be expected to have children of a high quality. On the other hand, those who move back to the farms are usually older, "disillusioned" (to use the expression of Dr. O. E. Baker, of the Department of Agriculture), and perhaps not too successful. According to many students of the problem, this vast internal migration takes the most energetic and intelligent to the cities where they become relatively sterile, while their

NUMBER OF CHILDREN UNDER 5 YEARS OF AGE PER 1,000 WOMEN 15 TO 44 YEARS OF AGE ON APRIL 1, 1930, URBAN COMPARED WITH RURAL POPULATION IN UNITED STATES



EXTREMES OF POPULATION GROWTH AND DECLINE

Figure 6

This graph gives some indication of the effects of rural and urban environment on the fertility of the American stock. The farm population of Leslie County, Kentucky, which represents near the maximum in fertility at the present time in the United States, is the same stock as are the seven "American stock" cities. In the one case we have a group scarcely over half maintaining itself, while in the other case the reproductive rate is high enough more than to double the population in a generation. Such extreme differentials in fertility almost certainly will have a profound effect on the inborn qualities of future generations.

less capable cousins on the farms furnish our chief reservoir of population growth. Also from an economic point of view, this vast movement of people creates serious problems concerning which Dr. Baker[44] is extremely pessimistic at the present time. Between 1920 and 1929, he says, this movement represents a contribution in education, living expenses, etc., of about $35,000,000,000 from the farms to the cities. When the present distribution of wealth is taken into account this huge "invisible tax" assumes even more ominous proportions.

Both in the United States and in England it has been found that country children have lower intelligence scores than city children. In general, say Lorimer and Osborn,[45] lower intelligence scores are more closely associated with rural environment than with urban — and in some remote rural districts (which generally have the highest birth rates) very low scores are frequent. They then present studies made in Michigan, Indiana, and Kentucky which indicate that children in the country fall behind urban children by as much as ten points in intelligence scores. These results are also supported by the army intelligence tests of large numbers, and also by intelligence tests made in occupational groups—farmers on the average having I. Q. scores just above unskilled laborers, which group shows the lowest scores among occupations. The alarming implications of this situation, when we consider the continued drain by the city of the most active young people, are fairly obvious.

In the light of these studies it is interesting to note that our urban population is failing by thirteen per cent to reproduce itself while the rural-farm population over-produces by as much as 62 per cent, according to Dorn and Lorimer, of the National Re-

sources Committee.[46] In short, our farm population has a reproductive rate about twice that of the urban inhabitants—and if the unskilled laborers in the cities were excluded, this difference would be very much larger.

From this quick survey of scientific evidence available, it seems that there is room for improvement in *quality* of American population growth. As regards *quantity*, it appears that we are approaching, if indeed we have not already passed, that "optimum number" which is conducive to the best interests of all the people. If we prefer to lower our standards of living to those of European or Asiatic countries, no doubt we "*can* hold" and "*can* support" a considerably larger population. But can we support a much larger number gracefully or even comfortably? Population pressure has European countries at each other's throats over resources, raw materials, and land, while in Asiatic countries, living too near to the subsistence level creates havoc whenever there is a great natural disaster such as a flood or a drought.

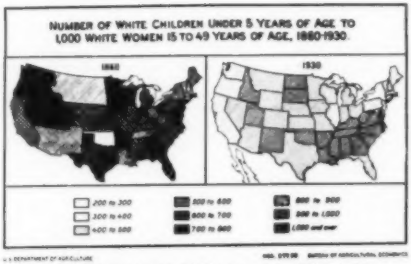
History teaches that as *large* nations become increasingly populated, economic, social, and political problems become highly complex. President Roosevelt has said, in his second Inaugural Address, that "nearly all of us recognize that as intricacies of human relationships increase, so power to govern them must also increase . . ." Twelve years before these words were spoken, and when this country contained some 15,000,000 fewer inhabitants, Professor A. B. Wolfe, eminent American population authority, reminded us that "increasing density means diminution of freedom, more intricate and expensive organization, loss of community self-reliance, and the multiplication of centralized and peremptory controls."[47]

In spite of such warnings by American scientists, some European statesmen and scientists appear to believe that the United States should have more immigration and a faster growth of population. (For examples see Kuczynski: *Population Movements*, pp. 73-76; Carr-Saunders: *World Population*, p. 219.) We are told that after the European countries have gone to all the expense to rear and educate their prospective emigrants the United States should be only too glad to welcome them. One answer to this alluring proposition was given by Dr. Charles H. Wickens, director of the Federal Statistical Office of Australia, when he said at the World Population Conference in 1927 (Proceedings, p. 323) that "the quality of the immigrant, like the quality of our dried fruit or our boots, may be excellent, but, if there is no market, they constitute a glut with all the trouble which a glut signifies in markets elsewhere."

As regards the effects of immigration on the birth-rate, we are sometimes told that immigrants help us lower our death rate and raise our birth rate. The records prove, however, that the foreign born in this country have higher death rates than the native born, and the census figures will show that we are not at present suffering from too few women in the childbearing ages. All population authorities know that our women are not exercising one-third and perhaps not one-tenth[48] of their natural power to increase population. It is also agreed among most competent authorities (with the exception of Dr. Gini and a few others) that the cause of our low birth rate is not biological, but *economic* and *social*. It is further held that immigration has a tendency to lower the birth rate of the native born. There may be some dispute as to how much immigration lowers the native birth rate, but most competent authorities in this country are agreed that this does happen to an undetermined extent, especially when the immigrants are accustomed to a lower standard of living than the native born.

Stationary and Declining Populations

We are told further that our declining birth rate will make us a nation of old and feeble people. This might be true if our birth rate continued to decline indefinitely. As Dr. Kuczynski would say, with the present trend in our



A SEVENTY-YEAR CONTRAST
Figure 7

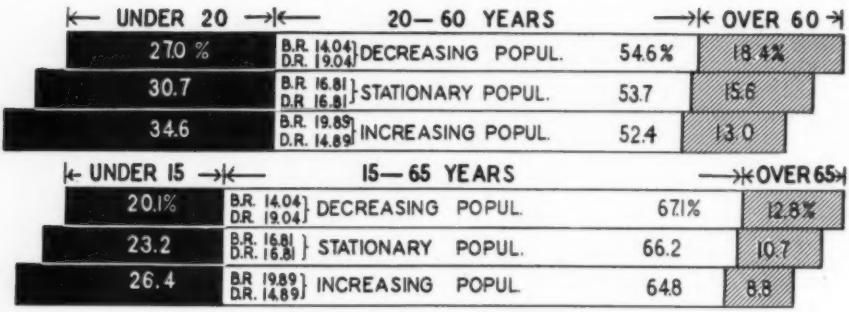
Since Civil War times reproductive mores in the United States have changed from multiplication to addition.

birth rate we are "bound to die out." The assumption or suggestion that our birth rate will continue downward is similar to 1932 stock market psychology, when it was believed that stocks would continue downward "indefinitely." Had the birth rate of the Colonial population continued, our numbers now would be about twice what they are, even without immigration. As a matter of fact, our birth rate already shows signs of ceasing its sharp decline and during the past few years has actually shown an increase over 1933. This support to the birth rate has not come from the foreign born because the large numbers of foreign born women who came to this country during the heydey of immigration, before the World War, are now past the reproductive age or the peak of the reproductive age, and their birth rate is falling faster than that of native women.[49]

It is true that for some time we shall have an increasing proportion of people between the ages of 60 and 80 years (the proportion beyond the age of 80 is relatively insignificant in either a growing, stationary, or slowly declining population), but this does not mean that the people in the productive age groups will be overburdened by a growing group of dependent old people, as many persons have been led to believe. As a matter of fact, as our population slows down and ceases to increase when we reach approximately 150,000,000 about 1980, we shall have fewer dependents as far as age is concerned than we have at present, even if persons between the ages of 40 and 65 were considered only half as valuable from a productive point of view as persons between the ages of 20 and 40 years.[50]

As the proportion of our "dependent old people" between the ages of 60 and 80 years increases the proportion of our dependent young people between the ages of 1 and 20 years will decrease much faster, and consequently we shall have a larger proportion of persons in the productive ages, and a smaller proportion in the dependent ages. The fact that we shall have a smaller proportion of persons between the ages of 1 and 20 years may frighten some of us into imagining that we will not have enough young people to replenish those in the productive ages. This is a needless fear because in a stationary non-increasing population there is a continuous replacement of one age group by another. But suppose our population begins to decrease? Then there is less fear that we will not have a large enough proportion in the productive age groups. A decreasing population has a larger proportion in the productive ages from 20 to 60 than either an increasing or a stationary population.[51]

Perhaps no scientist in this country is as alarmed about our declining birth rate and the possibility of a declining population a few decades hence as Dr. O. E. Baker,[52] Senior Agricultural Economist of the U. S. Department of Agriculture. He grants that "a stationary population is probably the most desirable condition for one reason because a larger proportion of the population is in the productive ages—20 to 60 or 15 to 65 years of age—than in an increasing or decreasing population." But he fears that after we have reached a stationary population our birth rate will continue to fall, causing a declining population which will have serious economic, social, and political consequences. (In passing it might be noted that there is some doubt whether there is a larger proportion of the population in the productive ages in a stationary than in a



LESS DEPENDENTS IN A DECREASING POPULATION

Figure 8

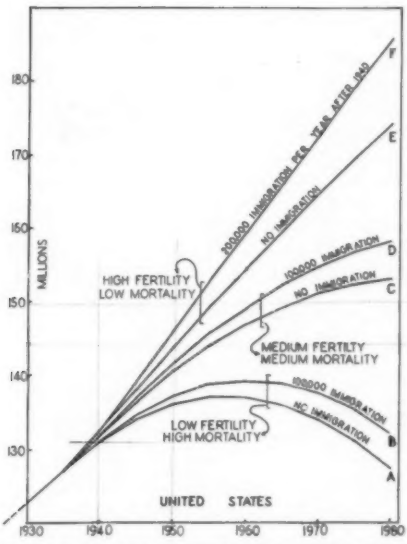
In a slowly decreasing population the proportion of individuals in the productive ages 15 to 65 years is greater than in a stationary population or in an increasing population. In a decreasing population the very young and the aged represent nearly equal numbers, while in a growing population youth exceeds age two to one. In a relatively long lived population such as that of the United States today (average longevity 61 years) the proportion of adults is very much higher than in earlier civilizations where the average age at death was as much as 20 years earlier than is the case today. Data for Male Population of U. S., 1930,—from Dublin and Lodka[81], page 122.

declining population [see Figure 8], but let us follow Dr. Baker further.)

He believes that births will continue to decline in this country because: (1) Our birth rate is following that of such countries as England and Germany and their birth rates are continuing to decline. (2) The lower classes will pattern their birth rate after that of the upper classes, which is now very low. (3) The youth of the nation is not interested in raising good-sized families. (4) There is a great migration of youth to cities where birth rates are low. (5) After 1950 there will be a decreasing number of potential mothers.

Furthermore Baker holds that a declining population will be unfavorable, if not disastrous, because: (1) It will cause a decline in the consumption of farm products. (2) The decline will be faster in the cities than in the rural districts unless rural-urban migration is accelerated, which will aggravate the loss of demand for farm products. (3) The farm population, which will not decline until after the city population, will spread to the poor crop land, causing degradation. (4) The decreasing proportion of young people and the increasing proportion of old people will bring about a wave of pessimism. (5) The birth rate of the upper classes will continue to decline ahead of that of the lower class, and cause a concentration of wealth in the hands of a very few people.

The writer is inclined to agree with many students of population that Dr. Baker is far too pessimistic about the decline in the birth rate for the following reasons: (1) Our birth rate need not follow those of England and Germany. Those countries are far more densely populated than we are. Furthermore, their populations have not begun to decline as yet. (2) The lower classes may pattern their birth rate after the present birth rate of the upper classes when knowledge of birth control becomes democratic, but this will remove an economic pressure on the upper and middle classes, which will then be able to support larger families at their accustomed standard of living. (3) The youth of the nation are not interested in raising good-sized families because economic opportunity is declining, due to the fact that the "open spaces" have been rapidly populated. A declining birth rate should give young men more elbow room and lessen competition for



POPULATION ESTIMATES DEPEND ON THE ASSUMPTIONS ONE MAKES

Figure 9

Estimates by Dr. P. K. Whelpton of population growth in the United States, 1930-1980, on the basis of various assumptions regarding birth and death rates and immigration. The effect of immigration on the birthrate of resident population might alter these curves. Dr. Whelpton believes that the medium fertility and mortality estimates probably will be most closely realized in fact. The high and low estimates indicate about the maximum or minimum in population growth or decline that is likely to occur under extreme conditions. Very likely present trends in vital indices will change materially before 1980. In proportion as we *guess* rightly regarding these trends, will we be able to claim to be good population prophets. Chart compiled from estimates presented by Whelpton.[60]

jobs. (5) Population growth is not dependent upon an increasing or decreasing number of potential mothers, but upon *economic* and *social* conditions. According to Pearl we are using only one-tenth of the childbearing capacity of our women.

Dr. Baker is too pessimistic as regards the effects of a declining population because: (1) A declining population need not cause a decline in the consumption of farm products. According to President Roosevelt one-third of our people are ill-nourished at the present time. Furthermore, it should be remembered that we shall experience an increase of a good many more millions of people before we begin to decline. In the meantime, wind and rain erosion, to which this country is particularly susceptible, may have encroached a great deal further upon our productive land. (2) When knowledge of birth control becomes more democratic the rural birth rate may decline faster than that in the cities. This should slow down the migration to the cities which has caused economic pressure and keen competition for industrial jobs—one reason why the urban birth rate is so low. (3) The farm population may not have to spread to poor crop land when a lower birth rate extends to rural areas. (4) The decreasing proportion of young people should make it possible for them to be healthfully supported and properly educated. This should not cause pessimism. And as we shall have a larger proportion in the productive ages—20 to 60 years—it should be easier for us to support dependent old people in a manner that should create optimism. (5) The assumption that the birth rate of the upper classes will continue to decline ahead of that of the lower classes is not borne out by recent developments in Sweden and in large German cities discussed below. This, and the fact that knowledge of birth control has been accompanied by a larger proportion of married people,[52a] makes for a more even distribution of children and wealth among the general population.

"Though there are transitional economic difficulties involved in adjusting production to an age distribution with fewer children and far more old people," say the experts of the *Study of Population Redistribution*, "there is no proof whatever that a somewhat smaller total population might not be just as near the hypothetical 'optimum' as that of the present day."[53]

This was written when this country contained about 128,000,000 persons. How much more true would it be after we increase about 25,000,000 more in population. While there will need be

adjustments for a stationary or slowly declining population, we shall have ample time to make them. This condition will not come upon us "like a thief in the night," says Dr. Baker, but "slowly . . . like the progress of the seasons." According to the Scripps Foundation for the Scientific Study of Population it will probably be 40 years before our population ceases increasing.

"A too rapidly declining population would obviously involve many severe problems," says J. M. Keynes, of Cambridge University. "But a stationary or slowly declining population may, if we exercise the necessary strength and wisdom, enable us to raise the standard of life to what it should be, whilst retaining those parts of our traditional scheme of life which we value the more now that we see what happens to those who lose them."[54]

The rather alarming decline of the American birth rate since the World War is not so unusual when we consider economic and social conditions in this country. After the Civil War the returning soldiers could go West to conquer new land from the Indians. But after the World War the returning soldiers found their jobs occupied by machines, women, and youngsters who had grown up in the meantime. For a time an artificial industrial prosperity in this country was stimulated by lending money to foreign countries to buy our goods, but the end of this unsound policy was to plunge us into the worst depression in our history.

The inhabitants of this country, without immigration, should increase about 25,000,000 persons during the next 40 years, according to the "medium" or conservative estimate of the Scripps Foundation for Research in Population Problems. In the light of the findings of the *Study of Population Redistribution* and the *National Resources Committee* this increase in numbers should be ample, if not more than ample, to supply this country with "needed" population.

The optimum or theoretical "right" number of people for this country will depend to a great extent upon the quality of the people. As Plato recognized two thousand years ago: "Like man, like state. . . . States are made out of the human natures which are in them." Whatever may be the temporary advantages of a policy of taking away from the successful to give to the unsuccessful, it does reverse the process of natural evolution and tends to encourage a faster growth of population among the unsuccessful and a slower growth among the successful, a process which must inevitably alter the character of a state if Plato's dictum is correct. If those least qualified either by heredity or by environment for a complex civilization continue to outbreed their more fortunate fellow citizens this paternalistic policy cannot long endure, and when the time of reckoning does come it may prove equally disastrous for the successful and unsuccessful alike.

Suggested Action

Since many scientists suggest that there is a "way out" we should perhaps present some of their recommendations. There is almost unanimous agreement among competent authorities that those who are definitely mentally defective should not reproduce their kind. This can be accomplished either by segregation or by voluntary sterilization. Efforts to segregate this element in our population are very expensive and have thus far proved only about one-tenth successful. Voluntary sterilization as an alternative or a supplement is far less expensive and more humane in practice since it permits many harmless defectives to be able to support themselves.

There is also almost unanimous agreement among objectively minded scientists that persons who are backward and mentally far below the vast majority of our people should be enabled to acquire the knowledge of family limitation which their more fortunate fellow citizens now possess and practice freely. As sterilization of defectives has been upheld by the United States Supreme Court, so a recent decision of the Federal Courts now leaves the way open for the under-privileged to possess medical knowledge as to ways and means of

controlling the size of their families. Third, it is agreed that if children of normal and superior individuals and groups can hardly be made economic assets at an early age, much can be done to ease the financial burdens of parenthood and to make an adequate progeny a social asset. Some of the economic burden can be removed through a changed social outlook, and through family allowances, etc. Such combined educational and economic changes may do much to encourage large families in these groups so that they will hold their own in the "Battle of Births," which in the last analysis determines what kind of a people we will become.

Perhaps it should be mentioned that there is some recent evidence that the difference in birth rates among economic and social classes (which became noticeable in Western Civilization about a hundred years ago) is diminishing. Broadly speaking, this difference in rates of reproduction appears to have been caused by three factors: (1) the demand for cheap unskilled laborers brought about by the industrial revolution; (2) the spread of knowledge of family limitation among the middle and upper classes; and (3) the advance in medical science and philanthropy in helping to equalize the death rates among economic and social classes.

There is reason to believe that during recent years these three causes of the differential rate of growth have been losing force. The machine has now developed to a stage where it is rapidly replacing unskilled laborers and the economic demand for them is diminishing. Second, knowledge of family limitation is now rapidly spreading to the lower economic and social classes. Third, while modern medicine and philanthropy still act to equalize the death rate among classes, they are also helping to spread knowledge of family limitation. The restriction of immigration to this country has hastened the operation of these forces in European countries. Lacking an outlet for surplus laborers through emigration, the lower economic classes in Europe are turning to means of restricting the number of their children. "The era of transoceanic emigration," says Professor Maurice R. Davie of Yale University, "is to be followed by an era of birth control."[55]

Birth Rates and Immigration Restriction

The steady decline in the birth rates of European countries since the enactment of immigration restriction laws by the United States and other "New World" countries appears to be bearing out Professor Davie's statement. When Premier Mussolini came into power in 1922 the annual birth rate of Italy was nearly 31 for every thousand inhabitants of that country. But, in the face of the Premier's strenuous efforts to raise the Italian birth rate, it steadily declined to about 23. As it is no secret that European countries contain more people than is conducive to the best interests of those people, when these countries could no longer export their surplus numbers a decline in the birth rate became an economic necessity. During the heydey of emigration the European countries which exported large numbers of emigrants could boast of birth rates higher than that of the United States—certainly a situation unwarranted by their natural resources compared with the resources of this country. Italy, one of the poorest countries in natural resources, actually raised her natural increase rate, births over deaths, 60 per cent during her mass emigration between 1880 and 1914. In the meantime, Dr. Kuczynski[56] tells us that the native born Americans in Massachusetts, which state was receiving large numbers of immigrants, were not even reproducing themselves. The migration operated in a manner to make room for more births in Europe and less room for births in this country, but when the migration privileges were withdrawn by the New World countries, the population problem took on a new meaning.

"Nothing has done so much in opening people's eyes to the meaning of the population question as the American immigration legislation," said Dr. G. W. Silverstolpe of the College of Commerce, Goteborg, Sweden, at the World Popu-

lation Conference in Geneva in 1927. "In my own country, Sweden, it has, within a short time, caused an almost complete change in public opinion on these matters. Nowadays very few people look upon birth control as an evil and nobody becomes really alarmed when the statisticians tell us that, with the prevailing rate of nativity, the population will, within a comparatively short time, show a state of stabilization.* * * The way of stabilization: that is the way all European peoples have to go—even all the nations—who wish to conserve that surplus of production over the bare necessities of life which makes culture possible."[57]

It was at this same conference that Dr. Carl Edin,[58] Department Chief in the Swedish Central Statistical Board, startled the scientific world by producing evidence that in Stockholm, where knowledge of voluntary parenthood had reached the lower classes, the birth rate of the upper social and economic classes was higher than that of the lower classes. This was a novel and unique thing in western civilization since about 1850 and it was difficult to believe. But five years later at the Population Union in London in 1932 Dr. Edin produced more evidence to support his earlier findings. In 1935, Dr. E. P. Hutchinson of Harvard University verified Edin's work.[59] In the meantime, Dr. F. Burgdorfer, of the German Statistical Office, found the same trend in the birth rate in large cities in Germany where knowledge of voluntary parenthood had also reached the lower classes. Burgdorfer found, as did Edin and Hutchinson in Stockholm, that in each income classification as the income became larger the size of the family also was larger. And what is of equal importance, he says: "I have recently been able to prove that today (1930) the number of children of families with large incomes is not diminishing but *increasing*."[60] This is rather remarkable considering the fact that the general birth rate of Germany was falling fast at that time.

Commenting upon the findings of Edin and Burgdorfer, Professor Warren S. Thompson, who is considered by many to be our leading population authority, says:

> It is not difficult to understand how this might be the case in populations where practically everyone knows about and practices birth control as he sees fit. These facts probably indicate new tendencies in population growth which are not unlikely to become general as the knowledge of birth control becomes universal among urbanized and industrial populations. The author has found evidence of a similar tendency in the low ratios of children to women among the Negroes in our northern cities.[61]

A trend in the birth rate similar to that found in Stockholm and in large cities in Germany seems to be beginning in New York City, the home of the birth control movement in this country. In studying the number of births to residents in that city's 305 health areas, Notestein[62] found that between 1933 and 1935 the number of births in groups that paid $60 per month and over for housing increased sharply, while the number of births in groups paying $45 to $59 per month declined. The number of births in those groups paying less than $45 per month declined even more.

Professor Ellsworth Huntington of Yale University reports from a study of Harvard and Yale graduates that not only do the most successful have the largest families but that "the size of the families is going back to such a level that these successful people are having more than enough children to replace themselves. . . . Birth control seems to promise the largest families in the best homes at all levels of society."

It is hoped that this review of population trends has given in hasty cross-section some of the more important conclusions and some of the present major battle-grounds of population theory. The attempt to study the problem of birth and growth and death with an objective technique similar to that of the astronomer proves in practice not to be easy. Vital statistics are really so vital in their implications that it is difficult always to view facts and trends objectively when the outcome of our calculations is not

the orbit of a distant planet but the prediction of the future life-history of our people. There are other weighty reasons why the experts do not always agree in their interpretations and we should bear these in mind when we consider the conflicts of opinion. The study of population trends by exact statistical methods is a very new science. Only in recent decades have even the most general data, such as total population, been available on any really adequate scale. Detailed data regarding births, migrations, deaths, etc., by classes of the population, are even yet not being obtained in adequate quantity. Furthermore, we appear to be living in a most abnormal time as far as population growth is concerned. The almost world-wide increase in population which began about 1620 is a unique phenomenon as far as the human species is concerned. Regarding the future developments of this "explosion of growth" we lack experience to make predictions. Thus there is an element of personal fear and hope in most of our writing about this subject which would be lacking were our knowledge greater. But there is no doubt that we know quite accurately the main *facts* of population trends. That the total population of the world is *increasing* no informed person seriously questions. That birth rates and death rates of European countries have been *declining* for several decades is admitted by all competent authorities. What *interpretation* is placed on these facts and on other correlated variables is to a greater or lesser extent a matter of opinion, and these opinions inevitably differ greatly.

We must remember, however, that the statistical data which are now being compiled with ever-increasing volume and accuracy, and the statistical techniques which are being applied to them, are a unique development in human history. Earlier civilizations seem to have reached an apex and to have declined without any exact information as to what birth and death trends were occurring among the people involved. Vital phenomena, such as the falling birth rate of Rome about two thousand years ago, were the cause of grave apprehension, but the interest in the subject arose almost after the event. We are rapidly bringing together exact statisical knowledge which is altogether unique, about what kind of people we are, and we are learning to guess with increasing accuracy what kind of people we must become if certain trends persist. Through studies such as those of differential birth-rates we are able to observe these trends very closely and we are given an opportunity to modify dangerous tendencies before it is too late. It remains to be seen whether we will have the intelligence, technical skill, and human sympathy and understanding to put this new knowledge to fruitful use in building the nations of the years to come. For the first time in human history the job *might* be done, and in that challenging possibility, *which has never before existed*, so far as we know, lies great hope for the future.

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◆

FIVE GENERATIONS IN NINETY-NINE YEARS

(*See Front Cover*)

In few human families are members of six generations living at one time. This unusual picture shows one-month-old Rosemary Bianca, of Indianola, Miss., and representatives of her ancestors for the five preceding generations. Her great-great-great-grandmother, Mrs. Mary Vincent (lower left) is 99 years old. Mrs. Theresa Mechapi, the great-great-grandmother, is 78, and the great-grandmother (Mrs. Grace Correno) is 59. The grandmother is 40, and the mother (Rosalie Bianca) is 20.

The length of generations varies considerably in different human families and races and under different climatic conditions. In northern countries the length of generation is placed at nearly a third of a century. In warm-temperate and tropical climates it is much less. The relative multiplication rate of human strains is tremendously affected by length of the generation. This baby has a total of 30 ancestors in five generations represented in this 99-year span, whereas with three generations to a century she would have only 14 ancestors at the end of the third generation. If we turn the matter around and consider the possible number of cousins little Rosemary might have, the geometrical nature of reproductive multiplication is strikingly evident. If we assume that 99-year-old Mary Vincent had four brothers or sisters and each of these sibs had had four children, who in turn each had four children, we find that the cousins of Rosalie Bianca (the mother) in the fifth generation would number 256. If all of these had four children, little Rosemary would in the course of a few years be one of a veritable regiment of 1,024 cousins, all of whom would call Mary Vincent great-great-great-great-grandmother. At the rate of four generations to a century baby Rosemary would have 256 cousins and at the rate of three generations to a century she would have only 64 under the same conditions. We have on the actual basis of this existing case a possibility that one couple would have sixteen times as many descendants as another in a single century as a result of a difference of about a third in the number of years which constitute a generation. In practice, of course, such reproductive consistency would never be encountered. Some lines would die out; some would be represented by only one child in one or more generations; some couples would have more than four children. Also the long-generation families might have more or less children than the short-generation-families, in which case the difference might be even greater than the Bryanistic sixteen-to-one ratio cited above.

The opposite tendency is reported on page 240, where an authenticated case of paternity at 97 (the mother being 29) gives nearly a century to one generation in the male line, but only 63 years per generation if the average age of the two parents is considered. This is over three times as long as the average for five generations in the female line of the Bianca family.—R. C.

A HEREDITARY EAR MALFORMATION

Transmitted Through Five Generations*

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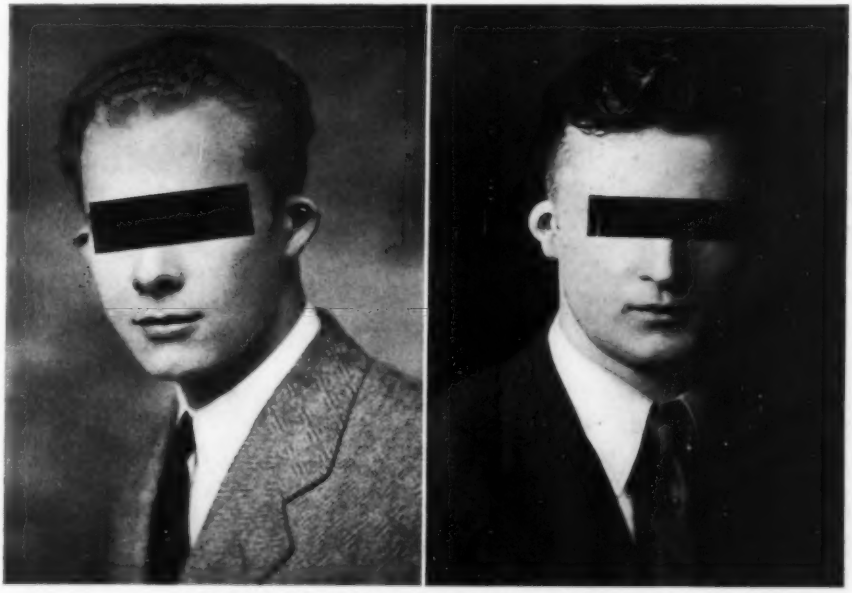


A DOMINANT GENE AFFECTING EAR FORM

Figure 10

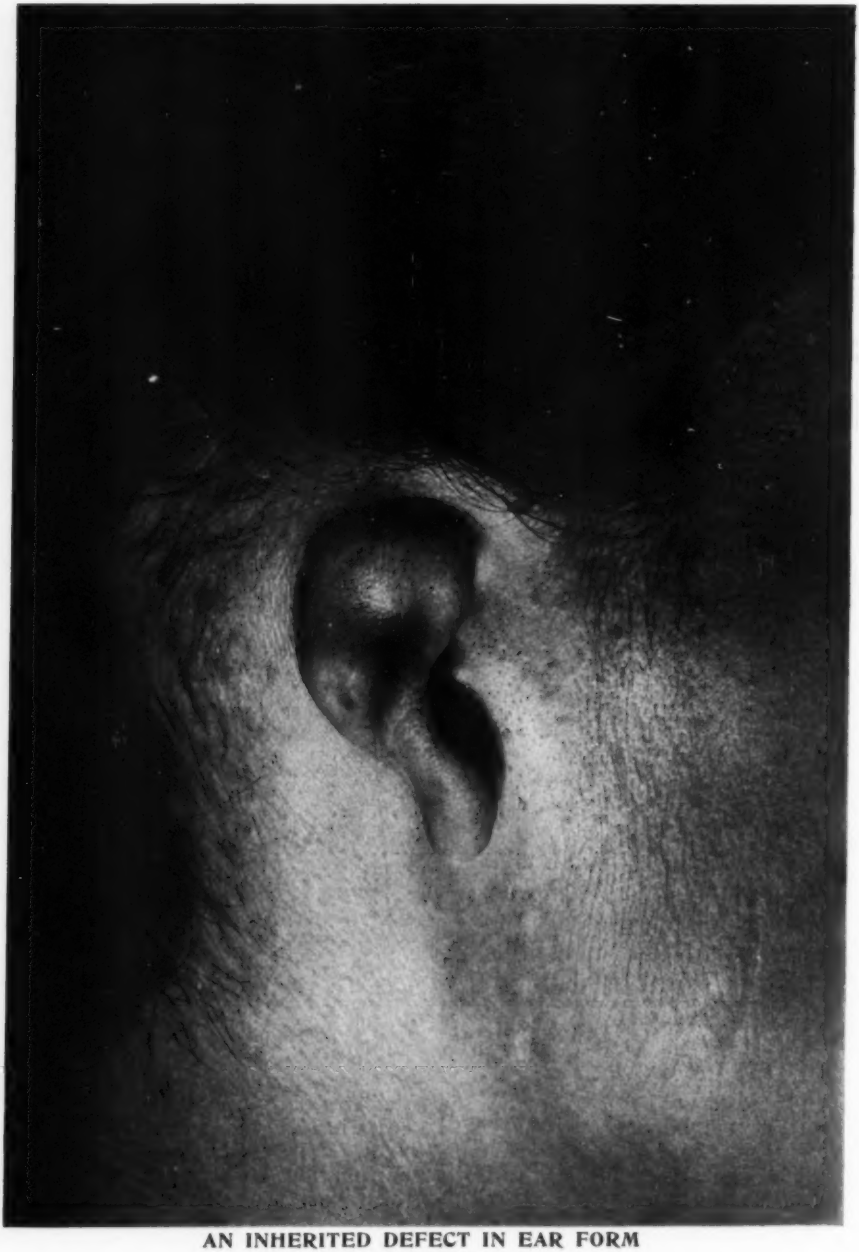
Two brothers (V-9 and V-10, Figure 11) of a family in which has been transmitted for at least five generations a gene affecting the form of the outer ear. On the average half the individuals showing this defect have transmitted it to their children, while normal sibs married to normal have not transmitted the defect.

ALTHOUGH many abnormally developed ears have been described and pictures of many varieties published, the cases discussed have usually been isolated instances of individual malformation and little information is available on the inheritance of such defects. The size of the family tree and the number of affected individuals here reported make it of considerable importance and interest.

A young woman exhibiting a congenital bilateral malformation of the ears presented herself for examination. A similar abnormality was known to have existed in her family for several generations. She wished to know whether she might expect to bear stigmatized children and whether her brothers and sisters without the defect might also transmit it.

The abnormality consists primarily of

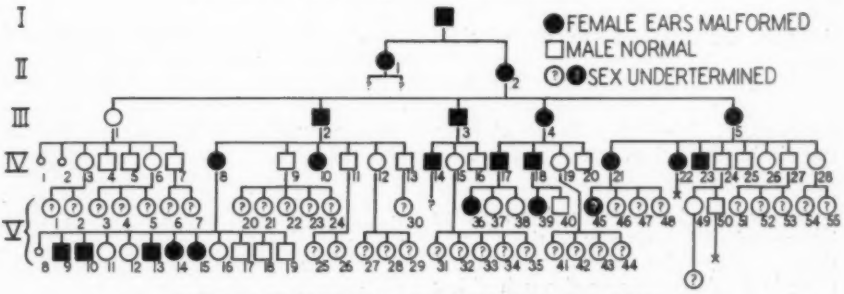
*This work was made possible by a grant from the Douglas Smith Foundation for Medical Research, The University of Chicago.



AN INHERITED DEFECT IN EAR FORM

Figure 11

Ear of propositus of this study (V-14), whose inquiry regarding the heritable nature of this defect led to the compiling of the accompanying pedigree (Figure 12). Instead of lying parallel to the side of the head, the ear has grown forward over the maetus to form a cup. The inner ear is normal, and no other manifestations of this gene have been noted.



FIVE GENERATIONS OF CUP-SHAPED EARS
Figure 12

Forty-five per cent of the fraternities in which 1:1 segregation for this character would be expected have malformed ears. All matings of affected individuals have been with normals. That the character is inherited as a simple mendelian dominant seems very likely in view of this pedigree.

a small, deformed, inverted pinna. The helix is short and extends downward instead of upward, the surface of the pinna normally directed toward the side of the head forms the lateral aspect of the ear and the lobule extends at right angles from the head. A cap-like structure is thus formed which completely covers the external auditory meatus when the head is in profile position. It invariably affects both ears. The internal ear is normal. No discomfort is suffered except for the psychic disturbance based on consciousness of the abnormality.

After considerable time a family tree embracing five generations and including 92 individuals was obtained. The most remote ancestor concerning whom information is available is the great-great-grandfather (I-1) of the patient. Nothing is known of his progenitors but he definitely suffered from the defect and transmitted it to his two daughters. Of the two children of one of these women (II-1) nothing is known. The other (II-2) married and had five children four of whom, two boys (III-2, 3) and two girls (III-4, 5) had a similar defect. The five children married, and each of the affected four transmitted the defect to some of the offspring. The one girl (III-1) of this generation who did not have the defect bore five children (IV-3, 4, 5, 6, 7), two girls and three boys, and had two miscarriages (IV-1, 2), none of the offspring exhibiting the defect. Of her children three (IV-3, 6, 7) are married and have a total of seven children (V-1, 2, 3, 4, 5, 6, 7), all of whom have normal ears.

The first boy (III-2) of the third generation had six children, two girls (IV-8, 10) with the defect, one girl (IV-12) and three boys (IV-9, 11, 13) without it. Of the four without the defect all are married, all have children (total number of eleven) and none have defective ears. One of the girls (IV-10), with the defect is unmarried, the other (IV-8) has eleven children, five of whom (V-9, 10, 13, 14, 15) suffer a similar abnormality. It is to this family our patient (V-14) belongs.

Among the other three individuals of the third generation one (III-3) had three children with one abnormal (IV-14), another (III-5) eight children with three (IV-21, 22, 23) showing the abnormality, and the remaining individual (III-4) had four children with two (IV-17, 18) defective.

Among the total individuals in the fourth generation there are eight who are defective, four males and four wemales, and 18 who are unaffected, eleven males, seven females, and two (stillborn) in which the state of the ears is unknown. In all cases the immediate parent was affected.

In the fifth generation there are a total of 55 individuals (including one miscarriage, in which the condition of the ears is unknown) eight of whom are affected. These include three males, four females, one sex unknown. They are the offspring of only four members of the fourth generation, these same four having also produced 13 normal children. The 12 members of the fourth generation who are married who did not

show the defect have 34 unaffected children.

Only three members of the fifth generation, all with normal ears, have thus far married. There is only one child in the sixth generation. It is normal.

Discussion

As far as is known there has been no intermarriage among any of the descendents of I-1. The defect is transmitted directly to the offspring through either the male or female parent.

There can be no doubt that the peculiar ears of this family depend on a single dominant gene. There is no relation to sex and any hereditary basis more complicated than a unit dominant factor would not persist undiluted for five generations. The differences in percentage incidence among the children of abnormals in different generations rest on such small numbers that they are probably merely accidental. The percentage for all generations (45.8 per cent abnormal) does not differ significantly from the 50 per cent expected of a dominant factor in backcrosses to the recessive normal. The fact that both ears are invariably affected in abnormals indicates regularity of expression. It differs in this regard from other known malformations such as polydactyly. This condition depends on a dominant gene that is highly variable in expression, often asymetrical and occasionally failing altogether to be manifest in "carriers," so that the character occasionally skips a generation.

From a study of her family tree our patient may expect to transmit ears which are abnormally formed to approximately one-half of her children. Her brothers and sisters who have similar ears will do likewise. Those who have normal ears will have children with normal ears and in succeeding generations the defect will not recur.

TABLE I. Number of individuals affected divided according to presence or absence of abnormal ears in parent.

		One parent abnormal			Both parents normal
Generation	A	N	Tot.	%A	N
2	2	0	2	100	–
3	4	1	5	80	–
4	8	13	21	38.1	5
5	8	12	20	40.0	34
6	–	–	–	–	1
Total	22	26	48	45.8	40

TABLE II. Total number of individuals with abnormal ears.

Generation	A	N	Tot.	%
2	2	0	2	100
3	4	1	5	80
4	8	18	26	30.8
5	8	46	54	14.8
6	0	1	1	0
Total	22	66	88	25

Summary

The malformation of the ears which exists in the described family is due to a single dominant gene. It is regular in expression and regular in transmission to half the children when marriage of abnormal to normal individuals occurs. There is no transmission by normal members of the family.

"Inheritance" of Avian Paralysis

Neurolymphomatosis gallinarium (Pappenheimer, Dunn and Cone, 1929) or avian paralysis is a widespread, transmissible disease of fowls characterized by infiltrations and tumor-like formations in the motor nerves and resulting in paralysis of muscles which control motion and other functions of the body. More or less related to avian paralysis is a group of diseases collectively known as leukosis.

* * *

The evidence indicates that the neurolymphomatous cell was transmitted from the hen and possibly from the rooster to the chicken through the egg. The exact method by which the egg is affected was not determined, although neurolymphomatous cells were found infiltrating ovules in affected hens and cells indistinguishable from them were found in the semen of affected roosters and the follicular fluid of diseased hens. The ovaries and testes of active transmitters were found at necropsy to be infiltrated with neurolymphomatous cells, and no hen or rooster was incriminated in this study as a transmitter that did not show such infiltration of the reproductive organs at some time.

* * *

During the course of these observations and experiments, evidence was secured which indicates that hereditary factors involving resistant and susceptible birds were found in certain forms of neurolymphomatosis, particularly in the infiltration of the ovules of the ovary and the nerve of the eye.—Gibbs, C. S., Observations and Experiments with Neurolymphomatosis and the Leucotic Diseases. *Mass. Expt. Sta. Bull* 337, Dec., 1936.

A PALE MUTATION IN THE GROUND SQUIRREL

An Albinoid Color-phase in *Citellus elegans* Resembling the Recessive Cream Mutation in the Rat

FRANK H. CLARK and WILLIAM L. JELLISON*

NORMAL AND "CINNAMON-BUFF" MUTANT SQUIRRELS

Figure 13

In a restricted area in Montana pale Wyoming ground squirrels frequently occur, though the form has not been noted elsewhere in the range of the species. The color closely parallels the recessive cream mutation of the black rat.

IN the population of ground squirrels, *Citellus elegans* (Kennicott) in Beaverhead County, Montana, there appears with some frequency a pale color phase. When observed in the field these rodents were first thought to be albinos. However, on closer examination they were found to have pigmented eyes and a distinctly tinted pelage.

According to Ridgeway's "Color Standards and Color Nomenclature," (1912) the mutant squirrels appear to be Pinkish Buff to Cinnamon Buff in color with very little difference between the color of the sides and back. The guard hairs as well as the fur hairs are buff in color and though tending to be uniform along their whole length are perhaps a little lighter at the base. The eyes seem to be normally pigmented.

Normal squirrels from the same area show the sides to be pale Smoke-gray blending into Drab-gray or Smoke-gray as one reaches the dorsal surface. With the exception of a narrow mid-dorsal streak which is almost Hair-brown in color, most of the dorsal surface is Drab-gray to Smoke-gray. A more detailed examination of individual hairs without reference to Ridgeway's standards reveals that the guard hairs are black throughout their length. The fur hairs which make up most of the pelage are black at the base, have a central light almost white band, and have the usual apical or sub-apical agouti band.

This "cinnamon-buff" variation in ground squirrels appears to be comparable to the recessive cream mutation in the black rat, *Rattus rattus,* and quite similar to a black-eyed, cream mutation in the meadow-vole, *Microtus pennsylvanicus,* which is being studied at the University of Michigan.

Of the 11 mutant squirrels seen during 1935 and 1936, 8 were captured alive and 1 was shot. A juvenile was captured on the Selway ranch 4 miles south of Dillon and another juvenile was taken along Birch Creek, 13 miles northwest

*Laboratory of Vertebrate Genetics, University of Michigan, and Assistant Parasitologist, United States Public Health Service respectively. The field notes were made by William L. Jellison, the color analysis by Frank H. Clark.

of Dillon. Although Wyoming ground squirrels are abundant in much of Beaverhead County and other parts of Montana, this color phase has not been observed except in the rather restricted area mentioned.

Immediately after capture it was evident that the mutant squirrels were more tractable than normal colored individuals and after a short confinement in the laboratory were quite docile.

GENES INVOLVED IN CANCER SUSCEPTIBILITY

To the Editor:

I do not know whether it is still permissible to join in the Cook *v.* Jaffe battle, but it appears to me that there has been an unfortunate tendency to follow Jaffe's bad habit of writing round personalities rather than round the facts upon which the various theories are based.

In the first place it must be realised that Slye has apparently abandoned the contention that cancer (irrespective of type and localisation) is inherited as a genetic unit. This was one of the principle bones of contention in the controversy with Little and was commented upon in the Editorial of the *Journal of Cancer Research*, Vol. 12 (1928).

Further it is necessary to realise the implications of any particular theory on the genetics of tumour formation. Roughly speaking there have been two tendencies in cancer genetics, that arising from the work of Slye and that arising from the work of Lathrop and Loeb. According to Slye's theories (or any modifications thereof) if two mice develop the same type of cancer they must be genetically similar as far as all pertinent genes are concerned. Basing our judgment upon the work of Lathrop and Loeb this need not be true. These workers found that strains of mice could be obtained having an incidence of mammary cancer that might take any value from 0 to 80 per cent. From this it would appear that we cannot make a confident statement upon the genetic constitution of an individual but must base our judgment upon a comparison of populations. It is at this point that Bernstein's calculations are of importance. He tested out the hypothesis of a single recessive gene for mammary carcinoma upon Lathrop and Loeb's data. First he selected a strain that was descended from a pair in which both parents developed mammary tumours and from a study of the tumour incidence in succeeding generations came to the conclusion that 77.4 per cent of homozygotes may be expected to develop cancer. Of this basis he pointed out that a strain with 9 per cent tumour incidence might possess 47 per cent heterozygotes. Further he studied the incidence of tumours in inbred generations which he considered originated in a cross between heterozygous dominant and recessive (Cc × cc). The observed tumour incidence was not inconsistent with the hypothesis that the original crosses had been of the type indicated above.

The essential point in the above argument is whether *in fact* the difference in cancer incidence shown between various strains can be accounted for in terms of differences in frequency of a single gene. If mating is strictly at random and there is no selection the relative gene frequencies will remain unchanged. In the presence of inbreeding and/or selection this equilibrium will be upset. By selection for and against cancer accompanied by brother-sister inbreeding Strong has been able to establish strains of mice in which the mammary tumour incidence varies from over 80 per cent (strain C_3H) to less than 10 per cent (strain CBA) intermediate values being attained by other strains. Opinions may differ as to the degree of heterogeneity to be expected in a strain after about 30 generations of brother-sister mating. Even if the most pessimistic estimates are accepted it is apparent that Bernstein's calculations can have no pertinence to Strong's results. Similarly when we turn to hybridisation experiments between highly inbred stocks we find that Lathrop and Loeb's results are confirmed, namely that the mammary tumour incidence in hybrids tends to resemble that of the maternal stock. Little and his colleagues have confirmed and extended this finding using strains that have been sufficiently inbred to preclude the possibility of any application of Bernstein's hypothesis. Further confirmation has been obtained by Korteweg in Holland with strains from Little's laboratory and by Pybus and Miller in this country with their own inbred strains. Other types of neoplasm have been less intensively studied than mammary tumours, but McDowall and Richter have shown that leukaemia shows a similar type of genetical behaviour whilst lung tumours and various types of non-epithelial growths seem to follow somewhat different laws. It may be that certain tumours will be found to depend upon the presence of single genes, these may be found to be recessive as is the case with Xeroderma pigmentosum or dominant as is the case with Von Recklinghausen's disease.

Lastly let me say that at this late date few (in possession of the facts) would dare to support the statement that cancer involves a complexity which differs in only one gene from the complexity involved in insusceptibility.

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ARE PLANT PATENTS "INVENTIONS"?

ALBERT E. FAY

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Mr. Fay's discussion, representing as it does the views of one who combines a professional knowledge of patent procedure with a continuing amateur interest in breeding, is a valuable addition to the views on this important subject expressed from time to time in the JOURNAL. *The suggestion of a "broad approach" to the problem of revising the Plant Patent Law in a workable direction is most interesting. The suggested wording does not seem to answer one need which certainly should be considered in any revision of the law, and that has to do with the possibility of including some form of varietal name-registration as a part of the patent. The advantages and disadvantages, as well as the legal complexities of such a plan need to be explored.*—EDITOR.

THE article "Plant Patent 110 Declared Invalid" in the JOURNAL OF HEREDITY for Oct., 1936, and the comments in the *Journal of the Patent Office Society* for March, 1937, have revived some thoughts which I have had for a long time on this subject. It is clear that the so-called act of invention is very different in the case of mechanical invention from that required in the case of the production of a new plant. In fact it is never or seldom that a new plant can be produced by any act of invention at all or by any inventive thought. The processes by which a new plant is produced, either by spontaneous mutation or by "planned patent inventions" are old. The results of these processes are natural and not artificial. It is difficult to see how they can constitute inventions in any case. But is it necessary that they should be in order to be patentable?

The Constitution empowers Congress to secure to authors and inventors the exclusive right to their writings and discoveries. Congress, R.S. 4886, has done this by authorizing the grant of a patent to "any person who has invented or discovered any new and useful art, machine, manufacture or composition of matter, or asexually reproduced any distinct and new variety of plant" etc. Congress obviously intended that such plants should be patentable. Are the courts going to throw them out because they are not inventions? Why does a new and useful discovery, otherwise conforming to the statute, have to be also an invention? The law, both before and after the Plant Amendment, authorized the patenting of any invention or discovery, etc. Are these two things or one? The courts seem to have treated them as one, requiring a discovery to be an invention also. But the Constitution authorized patents for discoveries without any special limitations, although it designated the discoverer as an inventor. Congress authorized the grant of patents for discoveries as well as inventions.

The courts have upheld many patents for discoveries which never required any act of invention, but were discoveries. But they have never admitted this obvious fact, apparently. Many such decisions can be used as precedents. If the courts insist on treating many chemical processes and products, discovered by the patentee and not invented, as inventions, well and good. It would seem, though, that eventually they must come around to the idea that such discoveries are patentable because they are discoveries and not because the word "invention" has been used in an unjustifiable sense for a long time by the courts in order to save a deserving patent. The courts have done violence to the English language many times for the same purpose. However, I claim that the Statute does not necessitate this in this case.

In the case in question all the "asides" (*obiter dicta*) were immaterial to the correct conclusion at which the court arrived and are not binding in future decisions. The idea that this particular plant could not be reproduced except from Horvath's original plant was presumably in the Bill of Complaint. The court practically had to accept it as far as finding infringement in that case. It was not true, however. Patent lawyers have made a business of writing plant patent specifications in the language they would have used if describing a rat trap or kitchen utensil. That is false representation. Their clients never went through any such process of reasoning as is described in many of the patents. It is easy enough to see this in a bud sport, but is equally true in a case like Horvath's. Presumably there was an accidental crossing and Horvath merely took one accidental plant and claims he bred it to a true variety. A cross once made producing a new plant certainly can be made again with the same result, but anybody who tries it may well have to have a great deal of persistence, or considerable luck. This would be infringement.

I once raised the "Scarlet Runner" or "Fire" bean. Among the plants I found one which had three of the five lobes of its corolla pink instead of scarlet. The other two were scarlet. This was a new blossom—to me, at least. The beans, instead of having a black and purple color, were a light greenish grey with the spots a little darker. The purple beans make a very dark mess when cooked, a fault that my beans had lost. As the bean is reproduced sexually only, I saved the light colored beans and kept the growing plants away from the scarlet ones for several generations and had my bean, after a few generations, breeding pure or nearly so. That was a discovery and in no sense an invention. I think the law ought to have protected it.

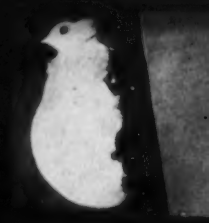
I think the same of the following. Blue Andalusian fowls, as is well known, breed 50 per cent blue, 25 per cent black and 25 per cent white or "splashed." I knew that there was a recessive blue "blood" in the white ones. I mated the latter with a Buff Orpington cock. Instead of breeding the F_1 generation in the usual way, back to one of the originals, I bred together the two of the F_1 generation coming nearest to what I hoped to get. In each generation I did the same. In about ten years I had a beautiful variety breeding at least 75 per cent true, and therefore admissible to the American Standard of Perfection, with an occasional throw back. Then I lost interest, proving that I was a typical inventor. My method of breeding was possibly a new one in a useful art and, in that case, might have been patentable under the old statute or the new. The product is not patentable, but when the law is changed it should be made so.

I suggest for the new Statute:

"Sec. 4886. Any person who has either invented or discovered any new and useful art, machine, manufacture, or composition of matter, or any new and useful improvements thereof, or who has either invented or discovered and reproduced any distinct and new breed or variety of plant or domestic animal or bird which breeds 75 per cent true, in cases sexually reproduced, not known or used" etc.

In this way, the statute could be made broad enough to cover, without argument, a large class of discoveries that cannot now be legally patented. Congress has intended to protect certain asexually reproduced plants, but the courts now may invalidate even this protection.

The idea of passing a new law, not based on the old one, providing for patenting plants seems less desirable than to put it all into one paragraph. If this proposed Statute is thought too broad, the part covering sexually produced plants and animals could be eliminated, but no good reason for that is seen.



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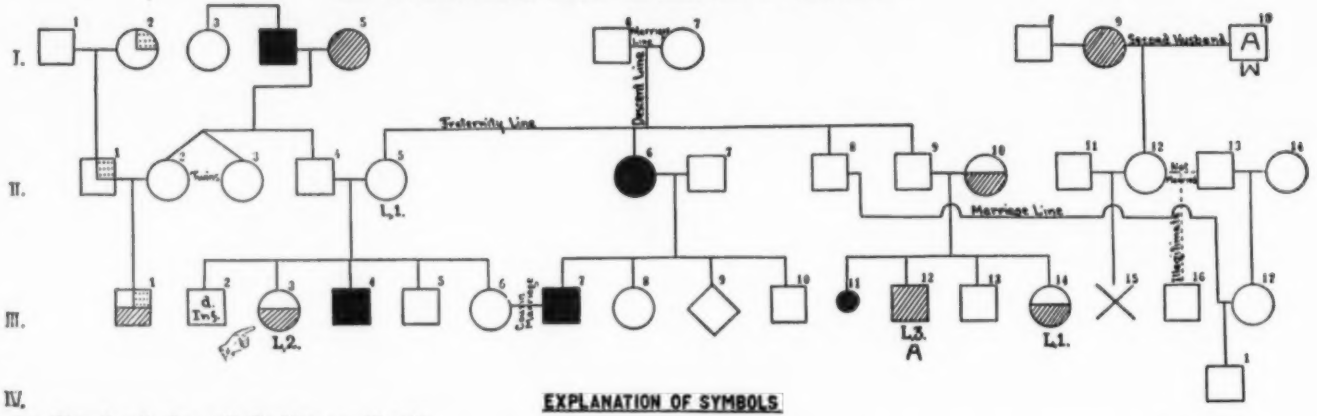
SAMPLE PEDIGREE CHART SHOWING THE MANNER OF CONSTRUCTION, AND THE USE OF STANDARD AND SPECIAL SYMBOLS.



EXPLANATION OF SYMBOLS

(a) **Standard for all Pedigree Charts:**

□=Male; ○=Female; ◇=Sex unknown; ●=Still-birth or Miscarriage; X=Children — number and sex unknown; ○○=Twins; d. Inf.=Died in Infancy; ☞ Points to the Propositus or central figure in the pedigree.

□—○=Parents.

□○□○○=Children.

Roman figures to the left indicate generations; Arabic figures locate individuals, (thus III. 7 is the young man in the third generation who married his cousin).

The following letters, placed in or around the individual's pedigree symbol, are standard for certain traits: A, alcoholic; B, blind; D, deaf; E, epileptic, F, feebleminded; I, insane, M, migraineous; N, normal in reference to traits under consideration; Ne, neurotic; P, paralytic; Sx, sexually immoral; S, syphilitic; T, tuberculous; W, wanderer.

(b) **Especially devised or selected for this particular pedigree-chart:**

■ ● = Successful leaders in politics. ◪ ◔ = Extra thumb on right hand. L.1. = Highly successful author. L.3. = Little or no ability in literary efforts. ▨ ◍ = Superior in vocal music. ▨ ◍ = Medium attainment in vocal music.

To fit the particular family and traits (whether physical, mental or temperamental; good or bad) under consideration, invent special symbols, or select special letters (in addition to those standardized under (a) above) to be placed within or near the particular individual's pedigree symbol, to indicate particular traits and their degree of development.

Prepared by the International Commission of Eugenics. See *Journal of Heredity* for August 1926. Additional copies may be obtained from the AMERICAN GENETIC ASSOCIATION.

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